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Bullous Lyme disease

To the Editor: A 57-year-old woman living in the Upper Connecticut River Valley presented in July 2011 with an acute blistering rash on her left thigh that she believed was caused by a spider bite. The lesion rapidly progressed over 4 days from a small tender red papule to a 12-cm annular, erythematous patch with a centrally located 5-cm tender, indurated bulla with surrounding vesicles (Fig 1). She also reported mild fever, headache, nausea, and arthralgia. She denied a history of a recent tick bite, hiking, or recent travel, but she did live in a wooded, rural area with numerous deer, frequently used a riding lawn mower, and stated she often saw ticks on her property.

The patient was seen by her primary care provider and in the emergency department before presenting to dermatology. Because of the bullous nature of the eruption, the differential diagnosis included bite reaction, cellulitis, herpes simplex, herpes zoster, and Lyme disease. Initial workup included complete blood cell count, basic metabolic panel, blood cultures, bacterial and herpes simplex/varicella-zoster virus cultures of the vesicular fluid, herpes simplex/varicella-zoster virus rapid stain, and Lyme IgG and IgM antibodies. All revealed normal findings. Given our confidence this was acute Lyme disease, we decided to treat the patient with doxycycline (100 mg by mouth, twice daily for 21 days). The rash and systemic symptoms rapidly resolved (Fig 2). Repeated Lyme antibody titers drawn 8 weeks later were positive for IgM by Western blot.

The first stage of Lyme disease usually features erythema migrans at the site of inoculation, accompanied by a flulike illness with nonspecific symptoms of fatigue, myalgia, headache, and fever. Of patients, 60% to 90% present with the classic erythema migrans rash, described as an erythematous macule that spreads centrifugally and may develop central clearing.[1] Atypical presentations have been reported, including hemorrhagic and necrotic cutaneous changes.[2,3] Vesicular or bullous variants of erythema migrans are rare, although 1 prospective study reported vesiculobullous changes of the central portion of the lesion in 8% of patients.[4]

Fig 1. Bullous erythema migrans in Lyme disease (initial presentation): 12-cm annular, erythematous patch with a centrally located 5-cm tender, indurated bulla with surrounding vesicles.

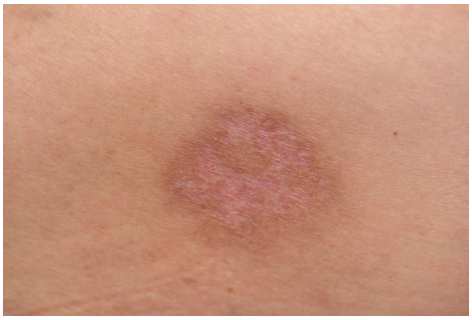

Fig 2. Bullous Lyme disease (2 months after treatment with doxycycline 100 mg by mouth, twice daily for 21 days): 5-cm patch of postinflammatory hyperpigmentation and hypopigmentation.

The recognition of erythema migrans is sufficiently distinctive to allow clinical diagnosis and treatment of Lyme disease. In fact, the visual inspection of the skin lesion is the preferred means of diagnosis as serologic testing is too insensitive in the acute phase of the disease (the first 2 weeks of infection) to be helpful diagnostically,[5] with IgM antibodies usually becoming positive 2 to 6 weeks after exposure.[6] This requires the physician to be aware of the classic appearance of erythema migrans as well as its multiple atypical variants, including bullous Lyme.

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Mid-dermal elastolysis as a manifestation of immune reconstitution inflammatory syndrome in an HIV-infected patient

To the Editor: The immune reconstitution inflammatory syndrome (IRIS) encompasses a group of disorders with different clinical manifestations occurring in HIV-infected patients after starting highly active antiretroviral therapy (HAART), resulting from the restored ability of the immune system to mount an antigen-specific inflammatory response. Previous studies have demonstrated that 10% to 25% of HIV-positive patients starting on antiretroviral therapy experience IRIS, and in more than half of them cutaneous involvement represents the first manifestation of the disease.[1]

A 32-year-old Caucasian man with a 13-year history of HIV infection (CD4 cell count 269 cells/mm^3, plasma HIV RNA load [VL] 16,315 copies/mL) started HAART in December 2012. Two months later his CD4 cell count increased to 465 cells/mm^3, and the viral load was undetectable. At the same time, the patient reported a progressive, asymptomatic, erythematous, parchment-like appearance of the skin on his trunk (Fig 1). He denied prolonged ultraviolet light exposure.

Fig 1. Mid-dermal elastolysis: asymptomatic parchment-like appearance of the skin on the trunk.

A skin biopsy specimen showed a sparse lymphohistiocytic infiltrate admixed with scattered, multinucleated histiocytic giant cells arranged between the collagen bundles in the mid dermis (Fig 2, *A*). Focal elastophagocytosis was also noted. Orcein staining highlighted a bandlike loss of elastic fibers in the mid dermis (Fig 2, *B*). A diagnosis of mid-dermal elastolysis was made. Four months later the patient experienced gastrointestinal symptoms associated with perianal fistulae. A diagnosis of Crohn's disease was confirmed by radiologic, endoscopic, and histopathological examination.

Mid-dermal elastolysis is a rare condition characterized by localized, bandlike loss of elastic fibers in the mid dermis.[2] It presents clinically with variably large areas of fine wrinkling, tiny perifollicular papules, or both. The pathogenesis is unclear. Several cases seem to be related to ultraviolet radiation, whereas in others immunologic mechanisms may be involved. The association with different autoimmune disorders and the elevated antinuclear antibody found in some patients with mid-dermal elastolysis have supported the hypothesis of an autoimmune origin.[2]

To our knowledge, mid-dermal elastolysis in the course of HIV infection has been reported only once.[3] In contrast to our patient, the onset of skin lesions was unrelated to treatment, and an association with actinic damage was suggested as